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Mortality from Copper Smelter Emissions Circa 1967

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Pope et al. (2007) found lowered monthly mortality rates during a 1967–1968 copper smelter strike, coincident with and attributed to widespread reduced airborne sulfate levels. The authors cited three "intervention" studies associating particulate emissions reductions with mortality reductions as supportive. Evidence below suggests that mortality reductions in the study by Pope et al. and "interventions" are likely linked to reductions in particulate matter (PM) types known to be harmful: high levels of biologically active metals and partially burned carbon.

The first "intervention" study (Pope et al. 1992), examined PM–mortality associations over 4 years, encompassing closure of a Utah steel mill (sulfate not measured; sulfur dioxide levels were "low"). Mortality rates were 40% greater than expected when the mill was operating, suggesting toxicity of mill emissions. Strongest associations were with respiratory disease, then cardiovascular disease. Filter extracts when the mill was operating contained high levels of lead, copper, and zinc and were more toxic (Frampton et al. 1999).

Mattson and Guidotti (1980) found women living in communities near copper smelters (1968–1975) in Arizona experienced highly elevated relative risks (RRs) for acute respiratory disease mortality: averaged RR for all six mining towns (40,000 combined population) was 5.61. Later, Small et al. (1981) found levels of arsenic, Cu, and Zn elevated by factors up to 100,000 in Arizona smelter plumes. Lead levels in plumes were comparable with those of other metals. Thus, Pb, Cu, and Zn levels were elevated when either the steel mill or copper smelters were operating, and acute mortality (especially respiratory) was elevated simultaneously.

Mortality associations with blood Pb have recently been found at low levels of Pb (Menke et al. 2006). Blood Pb has a half-life of about 1 month, reflecting current exposure; associations may indicate both chronic and acute effects (Schober et al. 2006)—relevant information for copper smelter emissions.

The second "intervention" study (Hedley et al. 2002) found mortality rate reductions following a mandated 1990 reduction of sulfur in residual oil and diesel fuels in Hong Kong. Later, Hedley et al. (2006) found that ambient vanadium and nickel were reduced up to 90%, concomitantly with reductions of sulfur in residual oil. Mortality or inflammatory associations with ambient residual oil emissions but not secondary sulfate were previously found (Grahame and Hidy 2004; Janssen et al. 2002; Maciejczyk and Chen 2005).

The third "intervention" (Clancy et al. 2002) found that mortality rates declined after uncontrolled domestic burning of coal was banned in Dublin, Ireland. Wintertime black smoke levels declined from 80 $\mu g/m^3$ before the ban to 20 $\mu g/m^3$ afterward; sulfate was not measured. Given the toxicology of partly burned hydrocarbons, mortality reduction would be expected.

The three "supporting" studies do not provide evidence that widespread secondary sulfate reductions were related to mortality reductions during the interventions. Rather, high levels of specific metals, or of black smoke, appear to have health relevance.

Toxicology suggests secondary sulfates per se are unlikely to be harmful at ambient levels (Schlesinger and Cassee 2003). Resolving this inconsistency requires researching mechanisms by which secondary sulfate or precursors are necessary to create a toxic mixture at ambient levels; for example, how much do which metals increase in solubility due to such processes, and how much harm occurs that would not otherwise occur? Either soluble or insoluble metals common to steel mills and copper smelter emissions can be harmful at high doses (Ghio et al. 1999). Research suggestions are available (Grahame and Schlesinger (2007).

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Mortality from Copper Smelter Emissions: Pope Responds

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Grahame's thoughtful and well-documented letter addresses one of the most important issues regarding our analysis of the mortality effects of a copper smelter strike in the U.S. Southwest (Pope et al. 2007). Grahame's basic contentions are that changes in exposure to secondary sulfate alone were not sufficient to explain the observed mortality effects, and that the mortality effects were more likely due to changes in exposure to co-pollutants, such as biologically active metals and black smoke.

My coauthors and I agree with Grahame regarding several points. As he argues in his letter and as we briefly discussed in our article, the copper smelter strike also resulted in changes in exposure to metals and other co-pollutants. There is certainly evidence that metals, black carbon, and other by-products of incomplete combustion and high temperature industrial processes contribute to the pollution's toxicity—as part of the complex mixture of fine particles.

We respectfully disagree in part with Grahame regarding the lack of evidence implicating secondary sulfate particles as contributing to adverse health effects. He asserts that the three "supporting" intervention

studies "do not provide evidence that widespread secondary sulfate reductions were related to mortality reductions during the interventions." However, in addition to changes in metals, black smoke, and other co-pollutants, one thing that all three intervention studies had in common was substantive changes in exposure to sulfate particles. In Utah Valley (Pope et al. 1992), the steel mill (largely from its coke ovens) was responsible for over 75% of the valley's total sulfur oxide emissions. During wintertime temperature inversions, high concentrations of fine particulate matter with a relatively high proportion of sulfates occurred. The closure of the steel mill resulted in a disproportionately large drop in exposure to both metals, sulfates, and other mill-related pollutants.

Mortality reductions in Hong Kong were also associated with reductions in sulfur oxide exposure (Hedley et al. 2002). In Dublin, although sulfates were not measured, the banning of bituminous coal certainly resulted in an abrupt reduction in particulate pollution—including sulfate particles (Clancy et al. 2002).

In addition to the intervention studies discussed above, there is ample epidemiologic evidence that sulfate pollution, as part of complex mixtures, contributes to adverse health effects. For example, the Harvard Six-Cities Study (Dockery et al. 1993) and the American Cancer Society prospective cohort studies (Pope et al. 2002) of long-term air pollution exposure found both fine particulates and sulfate particles to be associated with mortality risk. A workshop of several research teams on source apportionment of particulate matter health effects found that the sulfate-related component of fine particles was most consistently associated with daily mortality (Thurston et al. 2005). The relative toxicity of sulfates per se and the additive or synergistic effects of related co-pollutants remains a matter of study and debate (Chen et al. 2006; Grahame and Schlesinger 2007). Nevertheless, epidemiologic studies of the adverse health effects of air pollution (Pope and Dockery 2006) have implicated fine particulate pollution from at least three general sources: coal combustion, high-temperature industrial processes, and traffic sources.

Overall, the literature suggests that sulfates—as part of mixtures of fine particles that include metals, black carbon, and other by-products of coal combustion, high-temperature industrial processes, and vehicle emissions—can contribute to adverse health effects. We reaffirm our conclusion that the results of our analysis of the mortality effects of the copper smelter strike "contribute to the growing body of evidence that ambient sulfate particulate matter and related air pollutants are adversely associated with human health."

The authors declare they have no competing financial interests.

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Smelters and Mortality

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Pope et al. (2007) provide results for reduced mortality during the 1967–1968 smelter strike in the U.S. Southwest. They ascribed mortality reduction to a decrease in ambient sulfate from the smelters. Although I found the thesis interesting, there is confounding that should be noted involving *a*) inconsistencies in state mortality relationships; *b*) the trace metal role, and possibly carbon exposure from the plant complexes; and *c*) ambiguities associated with SO_4 sampling.

The basis for the study by Pope et al. (2007) is a study by Trijonis and Yuan (1978), who analyzed the National Air Surveillance Network (NASN) SO_4 and visibility. They attributed improved visibility across the Southwest to SO_4 reduction during the strike. Ambient SO_4 includes SO_4 from oxidation of sulfur dioxide in air (secondary) and that emitted directly (primary). NASN data [e.g. U.S. Environmental Protection Agency (EPA) 1971, 1972, 1973] indicate a strike reduction in SO_4 (0.1–3.6 μg/m^3) at sites in the region (Trijonis and Yuan 1978; Table 16) and not Pope et al.'s uniform 2.5 μg/m^3. However, the accompanying association with non–weather-related visibility change is problematic (e.g., Hidy 1984).

If the smelter SO_4 was present regionally, exposure to concentration gradients of SO_4, SO_2, and metals would be expected with distance from the plants (Eldred et al. 1983; Malm et al. 1990). Pope et al. (2007) did not differentiate their results by distance from the smelters, but some information in their article is relevant because mortality is associated mainly with population centers (cities).

The risk estimates for New Mexico presented by Pope et al. (2007) in their Figure 6 (dominated by Albuquerque; the nearest smelter is 300 km south southwest) show high levels of mortality reduction in spite of an increase in annual average SO_4 between 1966 and 1967–1968 and a negligible reduction during the strike. The mortality reduction in Nevada is largest of the four states presented by Pope et al. 2007 (Figure 6), even though Reno and Las Vegas (population centers) are upwind of the smelters and are far distant over mountain ranges from the nearest smelter at Ely. The smallest risk change is in Arizona, but reductions in Utah are similar to those of New Mexico. However, note that the population centers in Arizona (Phoenix and Tucson) and Utah (Salt Lake City) are close to smelters.

The results of Pope et al. (2007) are further confounded by the fact that trace metals and carbon accompany the emissions from plant complexes (e.g., Leipold and Chow 1998; Small et al. 1981). Local exposure to smelter emissions involves primary SO_4 as well as SO_2 [the apparent SO_4 concentrations are biased high by a variable SO_2 filter adsorption artifact (e.g., Lee and Wagman 1966; Lipfert 1994)]. Emission reduction would reduce SO_4, including the bias from SO_2 adsorption; metals such as copper, lead, iron, cadmium, antimony, chromium, nickel, and arsenic; and possibly carbon. Distant exposure would be enriched in secondary SO_4 up to a point, followed by decline from atmospheric dilution and deposition. Pope et al. (2007) mentioned the metal–SO_4 linkage but did not explore it relative to the SO_4 theory. The combined exposure in sulfur oxides and metals from the smelters preset in aerosols from many sources adds further complexity to interpreting their results, including the differences in Salt Lake City and the Arizona cities.

Recent research suggests that a combination of primary SO_4 and metals from oil combustion, as well as carbon emissions from motor vehicles, may be important factors in mortality risk (e.g., Grahame and Schlesinger 2007). The smelter inferences appear inconsistent with these findings.

Pope et al. (2007) focused on the strike in the Southwest. However, they excluded the same period in Montana as a cross-check on their results. In the 1960s, a major copper production complex was located in Anaconda, Montana. NASN data from nearby Helena and Glacier National Park do not show a significant change in average SO_4 concentrations during the strike years (U.S. EPA 1971, 1972, 1973). This appears inconsistent with the widespread reductions seen in the Southwest. This difference could be a valuable adjunct to their results if mortality data are available.

Pope et al. (2007) provided a "natural" experiment in regional sulfur oxide (and metals) reduction. Their results should be examined further to insure that their interpretation is robust.

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Smelters and Mortality: Pope et al. Respond

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Hidy makes several useful comments regarding our analysis of the mortality effects of a copper smelter strike in the U.S. Southwest (Pope et al. 2007). Regarding issues of atmospheric chemistry, the ambiguities of SO_4 sampling, and the role of smelter-related trace metals and carbon, Hidy is a well-respected expert, and we do not quibble with these comments. In fact, we briefly addressed the issue of accompanying metals in our discussion, and we are in general agreement that metals, in addition to sulfur oxides and other smelter-related air pollutants, might have played a role in the observed mortality reductions.

With regard to the epidemiologic evidence, one must be careful not to over interpret the small differences in state-specific estimates of strike-period reductions in mortality. A primary statistical inference illustrated in Figure 6 of our article (Pope et al. 2007) is that similar and consistent (not significantly different) mortality decreases were observed across all four Southwest states.

Available data also suggest regional strike-related reductions in SO_4 concentrations. Based on summary data (Trijonis and Yuan 1978, Table 16), the average (and percent) decrease in SO_4 concentrations for the urban monitoring sites were 2.7 $\mu g/m^3$ (38%) for Salt Lake City, Utah; 2.3 $\mu g/m^3$ (51%) for Las Vegas, Nevada; 3.6 $\mu g/m^3$ (62%) for Phoenix, Arizona; 2.6 $\mu g/m^3$ (62%) for Maricopa county (near Phoenix); 3.4 $\mu g/m^3$ (67%) for Tucson, Arizona; and 0.1 $\mu g/m^3$ (2%) for Albuquerque, New Mexico. Even the remote sites of White Pine Nevada, Grand Canyon National Park, Arizona, and Mesa Verde National Park, Colorado (near the four corners of Utah, Arizona, New Mexico, and Colorado), observed 1.5 $\mu g/m^3$ (76%), 1.5 $\mu g/m^3$ (60%), and 1.1 $\mu g/m^3$ (57%) reductions in SO_4 concentrations, respectively. The only notable exception to the region-wide strike-related reductions in SO_4 concentrations is the negligible reduction in SO_4 concentrations in Albuquerque [as noted by Hidy and discussed in our article (Pope et al. 2007)]. Regarding Nevada, data from the Las Vegas and White Pine monitoring sites indicated strike-related reductions in SO_4 similar to those observed at other comparable sites in the region.

Although our analysis of the mortality effects of a copper smelter strike has clear limitations, its unique contribution relates to the relatively simple motivation and natural experimental design. A well-defined 8.5-month copper smelter strike in the 1960s resulted in abrupt, well-documented regional reductions in SO_4 concentrations and improvements in visibility (Trijonis 1979). Available mortality data indicate a significant strike-period decrease in mortality, even while statistically controlling for time trends, mortality counts in bordering states, and nationwide mortality counts for influenza/pneumonia, cardiovascular, and respiratory deaths (Pope et al 2007). The estimated reduction in mortality is consistent with what would be expected given the average reduction in ambient concentrations of SO_4 particles and estimated mortality effects from the relevant literature. For example, both the Harvard Six Cities Study (Dockery et al. 1993) and the American Cancer Society cohort studies of long-term air pollution exposure (Pope et al. 2002) reported similar mortality risks associated with fine and SO_4 particulate pollution. Also, comparable reductions in mortality were observed following the imposition of restrictions on the sulfur content of fuel in Hong Kong (Hedley et al. 2002) and the banning of coal burning in Dublin, Ireland (Clancy et al. 2002).

Finally, it is unclear how the issues addressed by Hidy are likely to confound the estimates of strike-period mortality reduction from a well-defined statistical or epidemiologic perspective. However, these issues do "confound" how we interpret the estimated strike-period reductions in a broader context and to what extent we attribute the observed mortality reductions to smelter-source SO_4 and related pollutants. We appreciate Hidy's contributions to our efforts to interpret these and related results.

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Environmental Tobacco Smoke: Incomplete Research or Author Bias?

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As primary author and co-principal investigator of the 16 Cities Study (Jenkins et al. 1996), I was disappointed to read the article by Barnes et al. (2006). Their methodology, relying solely on Internet-based searching of trial and hearing testimony, tobacco industry documents, and other information, was clearly incomplete and suggests poor investigatory skills and/or deliberate selection of data or information to support a preconceived notion of the complex processes involved with the conception, conduct, data reporting, and interpretation of what became known as the 16 Cities Study.

One example (there are many) of the errors presented by Barnes et al. (2006) is the paragraph on page 1895 devoted to highlighting the fact that in the Broin flight attendant class action lawsuit, Judge Robert Kaye ruled (mistakenly I believe) that I could not rely upon the 16 Cities Study in my testimony for that case. What the authors failed to mention is that in the Dunn-Wiley environmental tobacco smoke (ETS) trial in Muncie, Indiana, less than 6 months later (Dunn and Wiley v. RJR Nabisco Holdings Corp, et al. 1998), Delaware Superior Court 1 Judge Robert Barnet Jr. overruled a similar motion by the plaintiffs, and noted that he did not find the motion well taken. Did Barnes et al. simply miss that ruling in their search for information, or did they ignore it?

Probably the most egregious distortions of the facts lies in the claims of Barnes et al. (2006) that we failed to make the Occupational Safety and Health Administration (OSHA) or the public aware of RJ Reynolds' involvement in the field or analytical laboratory work done for the 16 Cities Study and/or tobacco industry sponsorship of the study, and that although we ultimately did publish papers regarding the impact of smoking restrictions on workplace exposures, those papers were "published long after the close of the OSHA proceedings, ..." (Barnes et al. 2006, p. 1896). Although the authors cited my 5 January 1995 OSHA testimony, they did not mention the ≥ 1,000-word explanation of the role of RJ Reynolds and Bellomy Research in the 16 Cities Study in my formal presentation to OSHA (OSHA 1995). Neither did they mention the discussions during my questioning regarding the work flow in the study or the pages of discussions regarding the contractual arrangements between Martin Marietta Energy Systems [Oak Ridge National Laboratory's (ORNL) prime contractor at the time] and the Center for Indoor Air Research, the study's sponsor. More importantly, in the first article on the 16 Cities Study (Jenkins et al. 1996; p. 475), a paragraph is devoted to the tasks and responsibilities of the three institutions (ORNL, RJ Reynolds, and Bellomy Research). On pages 480, 481, and 483, there is a detailed discussion of the work flow, quality control, and data inspection among the three institutions. Finally, on page 500, we acknowledged the funding source for the study and the contributions of key individuals from the other two institutions. Did Barnes et al. (2006) just miss the aforementioned discussions, or did they ignore them?

As to the issue of whether OSHA was made aware of the impact of workplace smoking in a timely fashion, the contention of Barnes et al. (2006) is false. In fact, as part of its deliberations, OSHA conducted expert workshops on exposure assessment, health effects, and ventilation beginning in September 1997. Both S.K. Hammond and I, along with others, were invited participants of the workshop on exposure assessment held at Johns Hopkins University on 12–13 September 1997. At that workshop, I provided a variety of analyses to the panel, including one concerning the impact of smoking restrictions on personal exposures to ETS in the workplace. That analysis was eventually published (Jenkins and Counts 1999). For Barnes et al. to claim that we did not make OSHA aware of the impact of workplace smoking restrictions on exposure before the close of the proceedings, when one of the authors was present at an OSHA expert workshop that was part of OSHA's "proceedings" where I presented the data analyses in question, is both astounding and false.

The 16 Cities Study stands as the largest and most representative study of personal exposure to ETS ever conducted in the United States. The methodology used was sound and the findings scientifically valid. Seven peer-reviewed papers have been published from its work, including one in *Environmental Health Perspectives*. If Barnes et al. (2006) disagree with the manner in which we organized or interpreted the data in those seven articles, I would point out that a flat-field version of our 16 Cities Study results database has been available to the public since at least 2000 through either the Sapphire Group (Gevecker Graves et al. 2000), or from the Oak Ridge National Laboratory's web site (Oak Ridge National Laboratory 2005). We have always welcomed fresh eyes on our data, and we are disappointed that Barnes et al. appear not to have taken advantage of its availability.

Science is about a dispassionate analysis of the facts—all the facts. All of the facts must be analyzed, even if they do not support a hypothesis or preconceived outcome of the analysis. Anything less can be considered poor science or, at worst, politics.

The author currently sits on the Board of Directors of the Institute for Science and Health, a nonprofit organization focusing on underresearched diseases, which has received unrestricted grants from tobacco companies; he also serves on the organization's Tobacco Science and Health Policy Advisory Council. In addition, the author has acted as a subcontractor to the Oak Ridge National Laboratory (which acts as a subcontractor to the University of Kentucky's Biomedical Engineering School, which is in turn funded for this work by a grant from the External Research Program of Philip Morris, USA). He is also involved in litigation regarding environmental tobacco smoke exposure.

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Environmental Tobacco Smoke: Barnes et al. Respond

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We are gratified that Jenkins does not dispute the central findings of our study (Barnes et al. 2006), namely, that *a*) the 16 Cities Study grew out of the tobacco industry's plan to block any Occupational Safety and Health Administration (OSHA) standard on secondhand tobacco smoke; *b*) RJ Reynolds Tobacco was the originator of the 16 Cities Study and exercised substantial control of the research at all times; *c*) Jenkins et al. (1996) combined exposure data from restricted and unrestricted smoking workplaces and compared exposure data in an inappropriate manner that produced results the industry could cite to support its claim that workplace secondhand smoke (SHS) exposures were low compared with household exposures during its efforts to defeat indoor smoking restrictions; and *d*) a proper analysis of the data Jenkins presented indicates that smoke-free policies would halve the total SHS exposure of those living with smokers and virtually eliminate exposure for most others, supporting the need for smoke-free workplaces [and the polar opposite conclusion of Jenkins et al. (1996)]. [Compare Figures 1 and 2 of our article (Barnes et al. 2006)].

The disagreement appears to be in how transparent or opaque these facts were to the reader of Jenkins' original article on the 16 Cities Study (Jenkins et al. 1996) and to OSHA.

In his letter, Jenkins ignores our Table 2 (Barnes et al. 2006), which contrasts the actual roles that RJ Reynolds and other agencies played in the design, conduct, and management of the 16 Cities Study compared with how these roles were described by Jenkins in his publications and direct testimony. We did not say that he did not disclose that he was working for the tobacco industry; we presented evidence that the disclosures in his articles did not completely reflect the role that the industry played in conceiving of and controlling the study. In addition, lengthy cross-examination of Jenkins during the OSHA hearings was required to reveal the extensive involvement of RJ Reynolds, and that revelation was incomplete (OSHA 1995).

We also would like to address a few other small points. First, far from "deliberate selection of data," we followed standard snowball methodology (Malone and Balbach 2000) for searching the industry documents; we identified > 500 relevant industry documents, as well as court records and the published literature, as a basis for our article (Barnes et al. 2006). We did analyze the full public 16 Cities data set when preparing our article, but we did not cite it because we were able to present our analysis based on summary results from the published articles (Jenkins and Counts 1999; Jenkins et al. 1996). We did not mention the Dunn–Wiley trial (Dunn and Wiley et al. v. RJR Nabisco Holdings Corp. et al. 1993) in Indiana because the ruling was on a motion to strike Jenkins' testimony because of procedural issues relating to disclosure of expert witness testimony in advance of trial, not a challenge to the conduct of the 16 Cites Study, as made in the Brion case (Dunn and Wiley et al. v. RJR Nabisco Holdings Corp. et al. 1998).

Nothing in Jenkins' letter contradicts our conclusion that he and his colleagues presented the data from the 16 Cities Study in a way that conformed to the stated objective of the Tobacco Institute's "OSHA Projects" to "encourage adoption of a ventilation standard and to discourage adoption of a smoking ban or of a standard that requires separate ventilation for areas where smoking is allowed" (Tobacco Institute 1991). Indeed, as noted above—and unchallenged by Jenkins—a proper presentation of the 16 Cities data [Figures 1 and 2 of Barnes et al. (2006)] shows that employees in "smoking workplaces" have significant levels of SHS exposure and that smokefree workplaces substantially reduce overall exposure to SHS. This conclusion remains important because the tobacco industry and its allies still rely heavily on the 16 Cities Study in opposing regulation of SHS exposures.

S.K.H. reviewed Jenkins' documents for OSHA during its hearing on SHS in the workplace in 1994–1995. S.A.G. testified on behalf of OSHA in the same hearing on other issues related to SHS. Neither has any current relationship with OSHA. R.L.B. declares he has no competing financial interests.

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Erratum

In the article "Concentrations of Urinary Phthalate Metabolites Are Associated with Increased Waist Circumference and Insulin Resistance in Adult U.S. Males" [Environ Health Perspect 115:876–882 (2007)] by Stahlhut et al., the title of Table 1 incorrectly referred to "serum" metabolite concentrations instead of "urinary." The correct title of Table 1 is "Mean and median urinary phthalate metabolite concentrations (μg/g creatinine): NHANES 1999–2002."

The authors regret the error.